DETAILED ACTION

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 8/17/2009 has been entered.

Applicants' arguments filed August 17, 2009 have been received and entered. Claim 100-101 are newly added that are generally directed to the elected invention. Claims 1, 7, 17, 22, 85-101 are pending in the application.

Restriction/Election

Applicants' election without traverse of Group IV, claims 1, 7, 17 and 22 filed 30 September 2005 in response to Office Action mailed 7 September 2005 was acknowledged and entered. Since the election was made without traverse, the restriction requirement was deemed proper and was made FINAL. It was noted that claims 2-6, 8-16,18-21 and 23-84 were withdrawn from further consideration pursuant to 37 CFR §1.142(b) as being drawn to a nonelected invention. However, during the prosecution of this application, applicants had re introduced claims 92-93 in amendment filed on 8/9/2007 that specifically recited previously withdrawn molecule (antisense and antibody). Accordingly, restriction requirement of claims of group I and II were rejoined along with elected invention of group IV.

Claims 1, 7, 17, 22, 85-100 and 101 are under consideration.

Withdrawn -Claim Rejections - 35 USC § 112

Claims 1, 7, 17, 22, 85-98 and 99 were rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. Applicants' argument that the instant application and data suggests some nexus between P-cadherin expression and hair growth is persuasive. Examiner would agree to the extent that one of skilled in the art would identify molecule that enhance or inhibit P-cadherin and test whether molecule is hair growth inducer or inhibitor. Since method steps merely require testing whether the molecule is function as hair growth inducer or inhibitor and does not necessarily require molecule to perform the biological function, instant enablement rejection is withdrawn.

New Claim Rejections - 35 USC § 112- New Matter

Claims 1 and 93 are newly rejected under 35 U.S.C. §112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter that was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art (hereafter the Artisan), that the inventor(s), at the time the application was filed, had possession of the claimed invention. 37 CFR §1.118 (a) states that "No amendment shall introduce new matter into the disclosure of an application after the filing date of the application".

Base claim 1 is directed to a method of identifying a hair growth inducer comprising: identifying a molecule that induces P-cadherin; and testing whether said molecule is functional as a hair growth inducer, while dependent claim 95 limits the molecule that specifically bind to P-cadherin. However, limitation of molecule that induces hair growth and specifically binds to P-cadherin is not supported by the disclosure. The instant specification while providing support for a method for identifying a molecule that modulates (inhibits) hair growth, wherein molecule specifically binds to P-cadherin, is silent on a molecule that induces hair growth; wherein molecule specifically binds to P-cadherin. Thus, at the time the application was filed, an Artisan of skill would not recognize from the disclosure that Applicant was in possession of a molecule that specifically binds to P-cadherin and function as hair growth inducer, as claimed.

MPEP 2163.06 notes: "If new matter is added to the claims, the examiner should reject the claims under 35 U.S.C. 112, first paragraph - written description requirement. *In re Rasmussen*, 650 F.2d 1212, 211 USPQ 323 (CCPA 1981)." MPEP 2163.02 teaches that "Whenever the issue arises, the fundamental factual inquiry is whether a claim defines an invention that is clearly conveyed to those skilled in the art at the time the application was filed...If a claim is amended to include subject matter, limitations, or terminology not present in the application as filed, involving a departure from, addition to, or deletion from the disclosure of the application as filed, the examiner should conclude that the claimed subject matter is not described in that application. MPEP 2163.06 further notes "When an amendment is filed in reply to an objection or rejection based on 35 U.S.C. 112, first paragraph, a study of the entire application is often necessary to determine whether or not "new matter" is involved. Applicant should therefore specifically point out the support for any amendments made to the disclosure". This is a new matter rejection.

Withdrawn-Claim Rejections - 35 USC § 102

Claims 17, 22, 92, 94, 97 and 99 were rejected under 35 U.S.C. 102(b) as being anticipated by Sprecher et al (Nature Genetics, 2001, 29, 134-136, online published on 9/4/2001, IDS). Applicants' arguments filed August 17, 2009 have been fully considered. Applicants' arguments that the biopsy material is not capable of exhibiting hair growth and therefore cannot exhibit a decrease in growth of hair are persuasive. Therefore, rejection is hereby withdrawn. Applicants' arguments with respect to the withdrawn rejections are thereby rendered moot. The claims are however subject to new rejections over the prior art of record, as set forth below.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 1 and 93 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for method of identifying a hair growth inducer comprising: identifying a molecule that induces P-cadherin; and testing whether said molecule is functional as a hair growth inducer, does not reasonably provide enablement for the molecule that specifically binds to P-cadherin and induces P-cadherin thereby function as hair growth inducer. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

In determining whether Applicant's claims are enabled, it must be found that one of skill in the art at the time of invention by applicant would not have had to perform "undue experimentation" to make and/or use the invention claimed. Such a determination is not a simple factual consideration, but is a conclusion reached by weighing at least eight factors as set forth in In re Wands, 858 F.2d at 737, 8 USPQ 1400, 2d at 1404. Such factors are: (1) The breadth of the claims; (2) The nature of the invention; (3) The state of the art; (4) The level of one of ordinary skill in the art; (5) The level of predictability in the art; (6) The amount of direction and guidance provided by Applicant; (7) The existence of working examples; and (8) The quantity of experimentation needed to make and/or use the invention.

The office has analyzed the specification in direct accordance to the factors outlines in *In re Wands*. MPEP 2164.04 states: "[W]hile the analysis and conclusion of a lack of enablement are based on factors discussed in MPEP 2164.01(a) and the evidence as whole, it is not necessary to discuss each factor in written enablement rejection." These factors will be analyzed, in turn, to demonstrate that one of ordinary skill in the art would have had to perform "undue experimentation" to make and/or use the invention and therefore, applicant's claims are not enabled.

The breadth of the claim embrace a method of identifying a hair growth inducer comprising identifying a molecule that induces P-cadherin; and testing whether said molecule is functional as a hair growth inducer, wherein the molecule specifically binds to P-cadherin.

The specification provides prophetic guidance about a method to identify a peptide molecule that is a modulator (i.e., enhancer, or inhibitor) of P-Cadherin and further, said molecule is also hair growth modulator (see page 9 and 10 of the specification). However, specification is silent with respect to testing a molecule that specifically binds to P-cadherin and induce P-cadherin thereby function as hair growth inducer. Prior to instant invention, Sprecher et al (Nature Genetics, 2001, 29, 134-136, online published on 9/4/2001, IDS) teaches that a mutation in the *CDH3* gene which encodes P-cadherin is the cause for the autosomal recessive disorder congenital hypotrichosis that is s characterized by hair loss, thereby implicating P-cadherin in hair disorder (See figure 2, abstract). It should be noted that Hirai et al (Development, 1989, Volume 105, Pages 271- 277, art of record) teach using P-Cadherin antibody that specifically inhibits P-Cadherin to show partial suppression of the growth of hair follicles in the surface epidermis and vibrissa follicles of cultured mouse upper lip skin (Fig. 4F). It is noted that neither prior art nor instant specification provide any guidance of a molecule that specifically bind to P-cadherin and induces P-cadherin thereby function as hair growth inducer. An artisan of skill would have required undue experimentation to make and use the invention without reasonable expectation of success. In conclusion, in view of breadth of the claims and absence of a strong showing by Applicant, in the way of specific guidance and direction, and/or working examples demonstrating the same, such invention as claimed by Applicant is not enabled for the claimed inventions commensurate with full scope of the claims. The specification and prior art do not teach screening a molecule that specifically binds to P-cadherin and also induces P-cadherin expression thereby function as a hair growth inducer.

New-Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 17, 22, 92, 94, 97, 99 and 101 are rejected under 35 U.S.C. 102(b) as being anticipated by Hirai et al. (Development, 1989, Volume 105, Pages 271- 277, art of record).

Claim Interpretation: Instant rejection is applied to the extent rejection applies to a method of identifying agent that inhibits P-cadherin and testing said agent in a model to determine if it is functional as hair growth inhibitor. As recited these claim merely require testing molecule that may or may not have intended biological effect. These rejections are not applied to the method of identifying hair growth inhibitor intended for cosmetics purpose.

Regarding Claims 17, 22 and 92, 94 and 100, Hirai et al identify P-Cadherin antibody that specifically inhibits P-Cadherin in the surface epidermis and vibrissa follicles of cultured mouse upper lip skin. It is noted that Hirai et al also disclose testing the cultured skin in absence and presence of a monoclonal antibody specific to P-Cadherin to determine the effect on hair follicle of the skin meeting the limitation of claim 17 (See Abstract, at page 271, column 1, line24 to column 2, line17; page 271, column 2, lines 37-47). It is noted that cultured pieces of the upper lip skin *in vitro* shows normal morphogenesis of the upper lip skin with extensive outgrowth of vibrissa follicle (see page 272, col. 2, last para.), while addition of monoclonal antibodies to E-cadherin or/and P-cadherin resulted in abnormal morphogenesis in the explants. The results show that the growth of hair follicles was partially suppressed in presence of inhibitor of P-cadherin (Fig. 4F) meeting the limitation of the claim 101. Since instant claims do not recite any specific polypeptide, a monoclonal antibody is considered a peptide molecule wherein the peptide being a monoclonal antibody specific to P-cadherin that specifically inhibits P-cadherin expression.

With respect to claims 97 and 99, Hirai et al disclose inhibition that is determined by microscopic examination of skin culture in the mixtures of P-cadherin specific and E-Cadherin specific antibodies and assessed by a decrease in P-cadherin expression (see abstract and figure 3). Accordingly, Hirai et al anticipate claims 17, 22, 92, 94, 97, 99 and 101.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

Claims 17, 22, 88-90, 94, 97, 99 and 101 are rejected under 35 U.S.C. 103(a) as being unpatentable over Sprecher et al (Nature Genetics, 2001, 29, 134-136, online published on 9/4/2001, IDS) or Hirai et al. (Development, 1989, Volume 105, Pages 271- 277, art of record), Crystal et al (US Patent 6159950, dated 12/12/2000).

Sprecher et al teaches that a mutation in the *CDH3* gene which encodes P-cadherin is the cause for the autosomal recessive disorder congenital hypotrichosis that is s characterized by hair loss, thereby implicating P-cadherin in hair disorder (See figure 2, abstract). Regarding Claims 17, 22, 92 and 94, Sprecher et al identify P-Cadherin antibody and then test P-Cadherin or E-cadherin antibody on the fresh frozen skin biopsies obtained from a patient and a control sample. Sprecher teaches that P-cadherin is expressed in a subset of epithelial cells involved in hair-shaft growth regulation in the hair follicle (see page 135, col. 3, para. 1). Thus, inhibition of P-cadherin expression by any test agent would imply that it would inhibit hair-shaft growth. As recited claim 17, require testing of agent that in functional as hair growth inhibitor, which is clearly embraced by the teaching of the cited reference. Since instant applicants do not claim any specific polypeptide, a monoclonal antibody is considered a peptide molecule wherein the peptide being a monoclonal antibody specific to P-cadherin specifically that inhibits P-cadherin. With respect to claims 97 and 99, Sprecher et al disclose inhibition of P-cadherin expression that is determined by microscopic examination of skin tissue in the mixtures of P-cadherin specific and E-Cadherin specific antibodies that are assessed by immuno fluorescence meeting the limitation of claims 97 and 99 (see figure 2).

Regarding Claims 17, 22 and 92, 94 and 100, Hirai et al identify P-Cadherin antibody that specifically inhibits P-Cadherin in the surface epidermis and vibrissa follicles of cultured mouse upper lip skin. It is noted that Hirai et al also disclose testing the cultured skin in absence and presence of a monoclonal antibody specific to P-Cadherin to determine the effect on hair follicle of the skin meeting the limitation of claim 17 (See Abstract, at page 271, column 1, line24 to column 2, line17; page 271, column 2, lines 37-47). It is noted that cultured pieces of the upper lip skin *in vitro* shows normal morphogenesis of the upper lip skin with extensive outgrowth of vibrissa follicle (see page 272, col. 2, last para.), while addition of monoclonal antibodies to E-cadherin or/and P-cadherin resulted in abnormal morphogenesis in the explants. The results show that the growth of hair follicles was partially suppressed in presence of inhibitor of P-cadherin (Fig. 4F) meeting the limitation of the claim 101. Since instant claims do not recite any specific polypeptide, a monoclonal antibody is considered a peptide molecule wherein the peptide being a monoclonal antibody specific to P-cadherin that specifically inhibits P-cadherin expression. With respect to claims 97 and 99, Hirai et al disclose inhibition that is determined by microscopic examination of skin culture in the mixtures of P-cadherin specific and E-Cadherin specific antibodies and assessed by a decrease in P-cadherin expression (see abstract and figure 3).

While Sprecher et al/ Hirai et al teach a method of identifying a hair growth inhibitor by identifying a molecule that inhibits P-cadherin and testing said molecule to determine if it is functional as hair growth inhibitor in a frozen skin biopsies or skin explants, but differ from instant invention by not explicitly testing P-cadherin in a mammalian model.

However, such was to one of ordinary skill in the art. For instance, Crystal et al teach a method for promoting hair or retarding hair growths by testing an agent in experimental model (see abstract). It is noted that Crystal et al teach hair loss is a naturally occurring process in mammals and common among healthy adult humans, while hirsutism is manifested as excessive androgen-dependent hair growth in women (col. 2, lines 10-37). It is noted that the agonists or antagonists of the HH signal transduction pathway or products of normal activation of the HH pathway are applied to mammalian skin either as protein preparations or via gene transfer technology known in the art (see col.6, lines 31-35). Furthermore, Crystal et al teach testing the effect of antagonist by supplying the agent to the skin in an amount sufficient to antagonize the HH signal transduction pathway in hair follicles and thereby retard the growth of or promote the loss of hair within the skin (see col. 5, lines 29-31).

Therefore, it would have been *prima facie* obvious for a person of ordinary skill in the art to combine the respective teachings by testing P-cadherin antibody disclosed by Sprecher et al/ Hirai by supplying the agent to the skin in an amount sufficient to antagonize the P-cadherin expression in hair follicles and thereby retard the growth of or promote the loss of hair within the skin, with a reasonable expectation of success, at the time of the instant invention. A person of skill in the art would have been motivated to test P-cadherin antibody as hair growth inhibitor as a matter of design choice, because Sprecher et al disclosed that mutation in the *CDH3* gene which encodes P-cadherin is the cause for the autosomal recessive disorder congenital hypotrichosis that is characterized by hair loss. Other limitation wherein said hair growth inhibitor is for man or a woman would be obvious to one of ordinary skill in the art because it well known in the art both man and woman suffer from hirsutism in view of teaching of Crystal. One who would have practiced the invention would have had reasonable expectation of success because Sprecher et al/ Hirai had already implicated P-cadherin in the hair disorder of human. Given that p-cadherin is necessary for the morphogenesis of the hair follicle and that the absence of its functional form, local inhibition of p-cadherin functions would be effective strategy for hirsutism treatment. Thus, it would have only required routine experimentation for one of ordinary skill in the art to test P-cadherin antibody/antagonist in the known methods to test whether P-cadherin is functional as hair growth inhibitor. Thus, the claimed invention, as a whole, is clearly *prima facie* obvious in the absence of evidence to the contrary. It should be noted that the *KSR* case forecloses the argument that a specific teaching, suggestion, or motivation is required to support a finding of obviousness See the recent Board decision *Ex parte Smith*, --USPQ2d--, slip op. at 20, (Bd. Pat. App. & Interf. June 25, 2007) (citing *KSR*, 82 USPQ2d at 1396) (available at http: www. uspto.gov/web/offices/dcom/bpai/prec/fd071925.pdf).

Claims 17, 91, 96 are rejected under 35 U.S.C. 103(a) as being unpatentable over Sprecher et al (Nature Genetics, 2001, 29, 134-136, online published on 9/4/2001, IDS), or Hirai et al. (Development, 1989, Volume 105, Pages 271- 277, art of record), Crystal et al (US Patent 6159950, dated 12/12/2000) as applied to claims 17, 22, 88-90, 94, 97, 99 and 101 above, and further in view of Reinhard et al (USP 20030194406, dated 10/16/2003, filed 5/31/2002, effective filing date 5/31/2001).

The references of Sprecher et al and Crystal have been described above and relied in same manner here. Although, Sprecher et al/ Hirai and Crystal teach a method of identifying a molecule that inhibits P-Cadherin and test whether said molecule is function as a hair growth inhibitor, wherein said molecule is an monoclonal antibody that binds specifically to P-cadherin, but differ from claimed invention by not explicitly teaching use of antisense to inhibits the P-cadherin expression.

However, such was known in prior art. For instance, Reinhard et al teach the production of specific "P-cadherin antagonists", particularly antibodies or antibody fragments, or small molecules that specifically bind P-cadherin, as well as ribozymes and anti-sense oligonucleotides that modulate P-cadherin expression (See para. 15). With respect to claim 96, Reinhard et al teach a variety of standard methods to detect specific effect uisng fluorescence, immuno histological or immuno precipitation methods (see para. 251).

Therefore, it would have been *prima facie* obvious for a person of ordinary skill in the art to combine the respective teachings of Sprecher et al/ Hirai, Crystal and Reinhard et al. by substituting P-cadherin inhibitor disclosed by Sprecher et al. with another such as antisense oligonucleotides that inhibits P-cadherin expression as disclosed by Reinhard et al in hair follicles and thereby retard the growth of or promote the loss of hair within the skin as disclosed by Crystal, with a reasonable expectation of success, at the time of the instant invention. A person of skill in the art would have been motivated to use any P-cadherin inhibitor including antisense oligonucleotides, as a matter of design choice to significantly inhibits P-cadherin expression, said design choice amounting to combining prior art elements according to known methods to yield predictable results. One who would have practiced the invention would have had reasonable expectation of success because Sprecher et al/ Hirai had already implicated P-cadherin in the hair disorder of human. Thus, it would have only required routine experimentation for one of ordinary skill in the art to test different P-cadherin inhibitor including an antisense using the known methods to test whether P-cadherin is functional as hair growth inhibitor. Thus, the claimed invention, as a whole, is clearly *prima facie* obvious in the absence of evidence to the contrary. It should be noted that the *KSR* case forecloses the argument that a specific teaching, suggestion, or motivation is required to support a finding of obviousness See the recent Board decision *Ex parte Smith*, --USPQ2d--, slip op. at 20, (Bd. Pat. App. & Interf. June 25, 2007) (citing *KSR*, 82 USPQ2d at 1396) (available at http: www. uspto.gov/web/offices/dcom/bpai/prec/fd071925.pdf).

Claims 1, 7, 85-87, 98 and 100 are rejected under 35 U.S.C. 103(a) as being unpatentable over Hoeffler et al (U.S. Patent application 20010048917, dated 12/6/2001, filed 3/9/1998), Sprecher et al (Nature Genetics, 2001, 29, 134-136, online published on 9/4/2001, IDS)/ Hirai et al. (Development, 1989, Volume 105, Pages 271- 277, art of record) and Crystal et al (US Patent 6159950, dated 12/12/2000).

The references of Sprecher et al/Hirai and Crystal have been described above and relied in same manner here. Sprecher et al teaches that a mutation in the *CDH3* gene which encodes P-cadherin is the cause for the autosomal recessive disorder congenital hypotrichosis that is s characterized by hair loss, thereby implicating loss of P-cadherin expression to hair loss in human (See figure 2, abstract). Crystal et al teach a method of testing an agent for promoting hair growths in an experimental model (see abstract).Thus, while Sprecher et al/ Hirai suggest a role of P-cadherin in hair follicle development, but differ from claimed invention by not explicitly teaching using molecule that modulate cadherin expression.

However, identification of agents that enhance or inhibit P-cadherin in modulating the skin phenotype was known to one of ordinary skill in the art. For instance, Hoeffler et al teach a method of identifying a candidate agent which has an affect on the phenotype, genotype of skin equivalents that are useful in cosmetic dermatology. It is disclosed that the agent may be administered topically or subcutaneously to the skin or skin cell (see para. 79, wherein hair follicles, melanocytes and sweat glands, are present on the skin (see para. 37 and 71). Hoeffler et al also teach that test agent may include any cosmetic, therapeutic or pharmaceutical including telomerase, genes, oligos, peptides, cadherins and wherein expression of E- or P-cadherin is increased and the affect is determined meeting the limitation of claim 98 (See para. 78).

Therefore, it would have been *prima facie* obvious for a person of ordinary skill in the art to combine the respective teachings by testing P-cadherin modulator disclosed by Hoeffler et al by supplying the agent to the skin in an amount sufficient to enhance or inhibit the P-cadherin expression in hair cells and thereby enhance or retard the growth of hair within the skin, with a reasonable expectation of success, at the time of the instant invention. A person of skill in the art would have been motivated to test P-cadherin antibody as hair growth inhibitor as a matter of design choice, because Sprecher et al disclosed that mutation in the *CDH3* gene which encodes P-cadherin is the cause for human hair loss. Other limitation wherein said hair growth inhibitor is for man or a woman would be obvious to one of ordinary skill in the art because it well known in the art both man and woman suffer from hair loss in view of teaching of Crystal. One who would have practiced the invention would have had reasonable expectation of success because Sprecher et al/ Hirai had already implicated P-cadherin in the hair loss of human. Given that p-cadherin is necessary for the morphogenesis of the hair follicle and that the absence of its functional form results in alopecia in human. Thus, it would have only required routine experimentation for one of ordinary skill in the art to test P-cadherin modulator using the known methods to test whether P-cadherin modulator that enhances P-cadherin is capable of enhancing hair growth. Thus, the claimed invention, as a whole, is clearly *prima facie* obvious in the absence of evidence to the contrary. It should be noted that the *KSR* case forecloses the argument that a specific teaching, suggestion, or motivation is required to support a finding of obviousness

See the recent Board decision *Ex parte Smith*, --USPQ2d--, slip op. at 20, (Bd. Pat. App. & Interf. June 25, 2007) (citing *KSR*, 82 USPQ2d at 1396) (available at http: www. uspto.gov/web/offices/dcom/bpai/prec/fd071925.pdf).

Claim 1, 95 is rejected under 35 U.S.C. 103(a) as being unpatentable over Hoeffler et al (U.S. Patent application 20010048917, dated 12/6/2001, filed 3/9/1998), Sprecher et al (Nature Genetics, 2001, 29, 134-136, online published on 9/4/2001, IDS)/ Hirai et al. (Development, 1989, Volume 105, Pages 271- 277, art of record) and Crystal et al (US Patent 6159950, dated 12/12/2000) as applied to claims 1, 7, 85-87, 98 and 100 above, and further in view of Wakita et al (Int. J. Cancer, 73, 432–439, 1997)

The references of Hoeffler et al, Sprecher et al/Hirai and Crystal have been described above and relied in same manner here. Although, Hoeffler et al, Sprecher et al/ Hirai and Crystal teach a method of identifying a molecule that induce P-Cadherin and test whether said molecule is function as a hair growth inhibitor, wherein said molecule is a peptide or a gene, but differ from claimed invention by not assessing the induction using immuno precipitation methods.

However, such was known and routinely used in prior art. For instance, Wakita et al teach a variety of standard methods to detect induction of P-cadherin expression including immunoblotting method (see page 433, col. 1, para. 3 and 435, figure 2b).

Therefore, it would have been *prima facie* obvious for a person of ordinary skill in the art to combine the teachings of Hoeffler et al , Sprecher et al/ Hirai, Crystal and R Wakita et al. to detect induction of P-cadherin by immuno precipitation and immuno-blotting as a matter of design choice, in a method of identifying a test agent, with a reasonable expectation of success, at the time of the instant invention. Said design choice amounting to combining prior art elements according to known methods to yield predictable results. One of skill in the art would have had a reasonable expectation of success in combining the teachings of Hoeffler et al , Sprecher et al/ Hirai, Crystal with those of Wakita because it was routine in the art at the time of filing to detect induction of P-cadherin by immuno precipitation and immuno blotting as taught by Wakita to screen test agent that induce P-cadherin expression and function as hair growth inducer. Thus, the claimed invention, as a whole, is clearly *prima facie* obvious in the absence of evidence to the contrary. It should be noted that the *KSR* case forecloses the argument that a

specific teaching, suggestion, or motivation is required to support a finding of obviousness See the recent Board decision *Ex parte Smith*, --USPQ2d--, slip op. at 20, (Bd. Pat. App. & Interf. June 25, 2007) (citing *KSR*, 82 USPQ2d at 1396) (available at http: www. Uspto.gov/web/offices/dcom/bpai/prec/fd071925.pdf).

Conclusion

No claims allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to ANOOP SINGH whose telephone number is (571)272-3306. The examiner can normally be reached on 9:00AM-5:30PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Peter Paras can be reached on (571) 272- 4517. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Anoop Singh/
Examiner, Art Unit 1632